
# The Critically Ill Patient with Structural Heart Disease Part I: Aortic Stenosis

**Key Articles**

Jentzer JC, Ternus B, Eleid M, Rihal C. Structural heart disease emergencies. *J Intensive Care Med*. Published online April 21, 2020:885066620918776.

**Background**

- Acute decompensated heart failure and cardiogenic shock are increasingly presenting to our emergency departments and admitted to the ICU.
- Cardiogenic shock is still most commonly the result of acute myocardial infarction or progressive cardiomyopathy, but structural heart diseases are an important subset of patients that must be on the clinical differential.
- Structural heart diseases include aortic stenosis (AS), aortic regurgitation (AR), mitral stenosis (MS), mitral regurgitation (MR), left ventricular outflow tract obstruction (LVOTO), and acute ventricular septal defect or rupture.
- Typically present with severe shortness of breath and/or hypotension that often responds poorly to standard medical management for acute heart failure.
- The most challenging part about managing patients with structural heart disease is that these lesions often progress gradually over time, but acute decompensation is more often a secondary consequence of an acute illness rather than the valvular pathology itself.
- This is a great review that discusses a number of these disorders, but for the purposes of this podcast we are going to focus on one of the most common valvular disorders – aortic stenosis.

**What goes wrong and why**

- Aortic stenosis is the second most common form of valvular heart disease
- **Pathophysiology:** Severe AS causes fixed obstruction to LV ejection, increased LV afterload leading to progressive diastolic dysfunction due to LVH, and eventual systolic dysfunction as the LV dilates over time.
- The majority of patients with cardiogenic shock due to AS have LV systolic dysfunction, and it can be challenging to differentiate hemodynamic compromise from severe AS versus other progressive cardiomyopathies
- **Most common etiologies of AS:** Calcification and degeneration over time, congenital bicuspid aortic valve, degeneration or thrombosis of previous prosthetic valve, and rheumatic heart disease
- Accuracy of physical exam is unfortunately limited
    - Patients may be tachycardic and cool extremities from poor cardiac output

- Pulmonary edema may or may not be present
- BEWARE: classic findings of systolic ejection murmur may or may not be present depending on the systemic hypotension and volume status of the patient

**Ultrasonographic physical exam**

- Because of the limitations of physical exam in these patients, *echocardiography* remains the cornerstone of diagnosis but usually requires a detailed exam that includes doppler and color imaging.
  - **PEARL**: Limited/point-of-care echocardiograms that are commonly performed in the ED will often miss AS as a cause of cardiogenic shock in the ED. Generalized assessments of LV function (LVEF %), RV function will not provide important clues about a patient with severe AS' underlying structural disease.
  - To Review, Echo criteria for Severe AS includes:
    - Aortic valve area (AVA) $\leq$ 1 $cm^2$
    - Mean AV gradient $\geq$ 40 mmHg
    - Aortic valve max velocity $\geq$ 4 m/s
    - ** Note: Patients with long-standing AS can develop "low flow" severe AS as their LV systolic function decreases over time. In these patients, the AVA is still < 1, but they will not have the high gradient/velocity due to low stroke volumes.

**Management**

- **BEWARE**: Application of standard therapies for Acute Decompensated Heart Failure and shock may be ineffective *or even harmful*.
- The *fixed* obstruction from severe AS results in markedly increased LV afterload, which can be further exacerbated by hypertension due to endogenous catecholamines in patients with acute distress.
  - Unfortunately, responses to vasodilators (such as nitroglycerin or nicardipine) can be highly variable – as these may address systemic vascular tone, but not the fixed stenosis.
- Management of the normotensive or hypertensive patient:
  - Often times, these patients can often be treated similarly to stable heart failure patients
    - Treat volume overload with LOW doses of diuretics
    - Hypertension: conservative vasodilators therapy, not at your usual dose for patients with acute cardiogenic pulmonary edema from systolic heart failure
  - NIV-PPV can be trialed for respiratory symptoms
- Hypotensive patient
  - Hypotension in patients with AS decreases coronary perfusion pressure, leading to myocardial ischemia and worsening myocardial dysfunction that may trigger a downward hemodynamic spiral
  - These patients are often *PRELOAD* sensitive due to the diastolic dysfunction from LVH that occurs over time

    - Start with a small 250 cc bolus, which will often improve hemodynamics
    - If patient continues to be hypotensive, early initiation of vasopressors is ideal as excessive IVF resuscitation can overwhelm the sick LV and lead to rapid pulmonary congestion.
        - Norepinephrine is a reasonable first choice, as it will increase afterload and can also support the LV with reduced systolic function.
        - If the patient has a thick LV and hyperdynamic, a non-inotropic pressor such as phenylephrine may be tolerated better

- Additional considerations
    - In patients with low flow AS (Low EF + AS) an inotrope trial with dobutamine should be considered (and may be preferred over epinephrine) as the patient's systemic vascular resistance may already be high.
        - NIV-PPV can be cautiously used
    - Early consultation with cardiology and cardiac surgery can be helpful, especially if there is any need for advanced therapies such as IABP or mechanical circulatory assistance devices.
    - Surgical interventions such Balloon Aortic Valvuloplasty can be considered as a bridge for unstable patients who fail medical therapy, but ultimately a Transcatheter Aortic Valve Replacement (TAVR) or surgical AV replacement.
    - The clinical goal is often to medically stabilize prior to surgical intervention, as the 30-day mortality rate of patients undergoing emergency TAVI remains high.
        - A recently published large clinical trial found that mortality was 8.7% in patients admitted with heart failure due to AS and 33.3% in patients with AS and shock.

**Summary**

- Aortic stenosis is a fixed cause of LV afterload that can cause patients to respond poorly to standard treatments of acute decompensated heart failure.
- Basic echocardiography in the ED can be misleading as simple assessments of LV/RV function cannot account for the severity of valvular disease
- Severe AS requires an AVA $\leq$ 1 $cm^2$, but can be either high or low flow depending on chronicity and LV function.
- Diuretics, fluid resuscitation, and vasodilators can have variable effects depending on the patient's underlying physiology, so use them with caution.